Eur J Vasc Endovasc Surg **29**, 363–370 (2005)
doi:10.1016/j.ejvs.2005.01.004, available online at http://www.sciencedirect.com on SCIENCE DIRECT®

Results of Endovascular Repair of Inflammatory Abdominal Aortic Aneurysms. A Report from the EUROSTAR Database

C. Lange,[1] R. Hobo,[2]* L.J. Leurs,[2] K. Daenens,[3] J. Buth[2] and H.O. Myhre[1]
On Behalf of the EUROSTAR Collaborators

[1]*University Hospital, Trondheim, Norway;* [2]*The EUROSTAR Data Registry Center, Eindhoven, The Netherlands; and* [3]*The University Hospital, Leuven, Belgium*

Objectives. *To investigate the results following endovascular treatment of patients with inflammatory abdominal aortic aneurysms (IAAA).*
Design. *Retrospective study based on the EUROSTAR registry.*
Material and methods. *Patients included in the EUROSTAR registry with IAAA (*n*=52, 1.4%) were compared to those having aneurysms without aortic fibrosis (*n*=3613, 98.6%). The mean follow-up period in patients with IAAA was 23 months (range 1–60). In 11 of the patients detailed information on the effect of endovascular repair and perianeurysmal fibrosis and ureteral entrapment was obtained by a dedicated questionnaire.*
Results. *Twelve patients (23%) with IAAA had preoperative impairment of renal function and five had known hydronephrosis. Variables that were significantly associated with IAAA included younger age (*p*<.0001, mean difference 5.9, CI 3.7–7.9) and lower pulmonary risks score (OR 0.38, CI 0.19–0.74). At completion of the endovascular procedure, device stenosis was more frequently observed in patients with IAAA (OR 18.1, CI 3.52–93.0). There were no differences with regard to the rates of mortality, rupture or conversion in patients with IAAA and controls. In the majority, the aneurysm size regressed irrespective of nature of aneurysm. Of the 11 patients with a detailed assessment three had deterioration of renal function and three still had ureteral entrapment during follow-up.*
Conclusion. *Despite persistence of perianeurysmal inflammation in a proportion of patients operative and midterm results of endovascular repair were comparable in the patients with inflammatory and standard AAA.*

Keywords: Abdominal aortic aneurysm; Inflammatory aneurysm; Endovascular repair.

Introduction

Five to 10% of abdominal aortic aneurysms have an inflammatory component (IAAA), characterized by a white glistening fibrotic surface, a thickened aneurysm wall and adhesions to neighbour structures. The thickened wall can be observed on CT and is usually in the range of 0.5–3 cm. Histologically, the muscular and elastic structures of the media are replaced by fibrotic tissue. Abundant lymphocytes and plasma cells are present. Patients with IAAA often have symptoms of abdominal or back pain. General symptoms like fatigue and weight loss are also common. The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are usually higher than in patients with abdominal aortic aneurysm without fibrosis.[1–3] The fibrotic changes may represent a difficulty during open surgery. This is reflected by a longer operating time, a higher mortality and morbidity and a greater need for blood transfusions when compared to non-inflammatory aneurysm.[4–6] Theoretically, therefore, endovascular repair (EVAR) could be an option in the treatment of IAAA, however, variation in outcome has been reported. In some cases, a successful result with shrinking of the aneurysmal sack has been observed.[7–10] In contrast, others have reported an increased inflammatory response following EVAR in these patients (Fig. 1).[11,12]

Since, the indication for EVAR in patients with IAAA remains controversial, the purpose was to investigate the outcome in patients with IAAA treated by EVAR and reported to the EUROSTAR register. The results were compared with EVAR performed in patients with non-inflammatory aortic aneurysm reported to the same register.

* Corresponding author. Roel Hobo, MSc, Department of Vascular Surgery, Catharina Hospital, P.O. Box 1350, 5602 ZA Eindhoven, The Netherlands.
E-mail address: eurostar@iae.nl

1078–5884/000363+08 $35.00/0

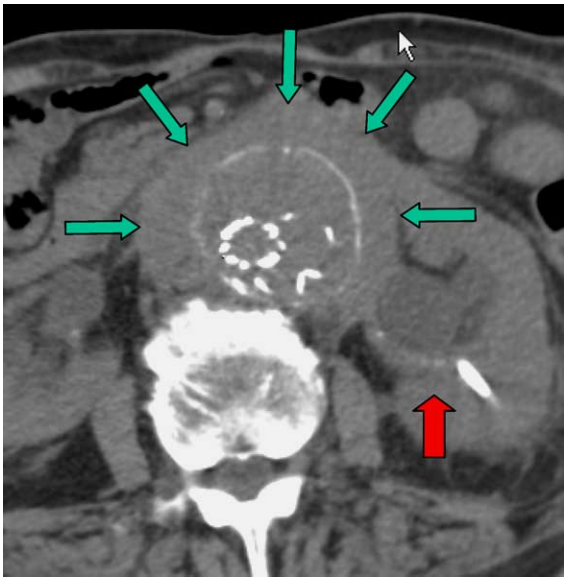

Fig. 1. CT examination 4 months after stentgraft repair of an IAAA. Note thick layer of perianeurysmal fibrosis (arrows). The left ureteric system became dilated after the procedure and needed drainage (larger arrow).

Material and Methods

This report summarises the experience collated in the EUROSTAR-database as of October 1996 to November 2003. The data of 3665 patients operated over a 7-year period until October 2003 constituted the basis of this analysis. The experience was obtained from 90 centers in Europe and the contributors to this series are listed in the Acknowledgement. The organisation of the EUROSTAR Registry and reports on various aspects after EVAR has been published previously.[13–15] All patients had a minimum follow-up period of 1 month. The mean follow-up period in patients with IAAA was 23 months (range 1–60). Patients with an aortic aneurysm smaller than 4.0 cm in diameter, including those with large iliac aneurysms, were excluded from this study cohort. An exception of this condition was IAAA, for which a diameter threshold for inclusion of 3.0 cm was used.

To assess the effect of IAAA on the early and mid-term outcome after EVAR the study cohort was subdivided according to the information provided on the case record forms (CRFs) about the inflammatory status of the aneurysm: patients with inflammatory aneurysms (IAAA) and patients with non-inflammatory aneurysms (non-IAAA). Specific details regarding the increase or decrease in the inflammatory response at follow-up were based on CRP, ESR and CT. Patients with IAAA were identified and their details derived from the free text fields in the CRFs as there were no queries specifically directed at IAAA. In addition, all centers participating in the EUROSTAR Registry received a letter requesting identification of patients with IAAA. To retrieve additional detailed data related to IAAA, a questionnaire was sent to the institutions whose records indicated endovascular treatment of patients with inflammatory aneurysms. Additional information regarding follow-up CT examinations and inflammatory serum markers for 11 patients were completed and returned to the EUROSTAR Data Registry Center.

Inclusion criteria as defined in the EUROSTAR registry protocol, comprised elective treatment of AAA with vascular anatomy suitable for the implantation of a stent-graft. Baseline data, including co-morbidity, estimate of unfitness for open repair, anatomic aspects and operative details were recorded by the participating institutions on CRF's and submitted for inclusion to the Data Registry Center. Findings at follow-up visits, which involved clinical examination, CT-assessment or (in 5% of the visits) angiography, MRI or ultrasound, were recorded in data forms and returned at regular intervals to the Data Registry Center for processing and analysis. There was no outside monitoring of the centers or involvement of a core laboratory for the evaluation of CT-scanning or other imaging studies. Follow-up visits according to the protocol were scheduled at 1, 6, 12, 18 and 24 months and annually thereafter. Reminders for overdue follow-up data were regularly sent to the participating institutions. Outcome was reported according to the guidelines from the Society for Vascular Surgery/American Association for Vascular Surgery (SVS/AAVS).[16] Deaths were classified as aneurysm-related or all-cause deaths.[17] The latter included death related to co-morbidity and conditions unrelated to the aneurysm. Aneurysm-related deaths included all deaths within 30 days and deaths that occurred as a result of aneurysm rupture, endograft infection or death within 1 month after a secondary surgical procedure for late complications of the aneurysm.

Other outcome events observed during follow-up included endoleaks, migration, severe device kinking, occlusion, stenosis and aneurysmal growth. Only endoleaks that were identified at 1 month and thereafter were included in the analysis, while endoleaks at the completion angiography were considered. Endoleaks were classified into type I, II and III as previously described.[18] In cases with different types of endoleaks observed at different follow-up periods, types I and III were considered above type II for the analysis. The interval between the date of surgery and the date on which the endoleak was identified for the first time, was used for the life-table analysis.

Table 1. Patient characteristics at the time of operation

	Inflammatory $N=52$	Non-inflammatory $N=3613$	p-value
Age (years, mean ± sd)	65.8 (±10.1)	71.6 (±7.6)	<.0001
Length of follow-up (months, mean ± sd)	22.8 (±18.2)	17.9 (±14.9)	.0205
Ratio male:female gender	96.1:3.9	94.4:5.6	.59
ASA classification III, III$^+$/IV	25 (48.1%)	1875 (51.9%)	.58
ABI ≤ 0.87*	2 (9.1%)	385 (20.6%)	.18
Diabetes	5 (9.6%)	418 (11.6%)	.66
Smoking	19 (36.5%)	817 (22.6%)	.0175
Hypertension	24 (46.2%)	2272 (62.9%)	.0133
Hyperlipidemia	14 (26.9%)	1438 (39.8%)	.06
Cardiac disease	23 (44.2%)	2146 (59.4%)	.0272
Carotid artery disease	8 (15.4%)	551 (15.3%)	.98
Renal insufficiency	12 (23.1%)	666 (18.4%)	.39
Reduced pulmonary function	11 (21.2%)	1497 (41.4%)	.0032
Previous laparotomy	17 (32.7%)	967 (26.8%)	.34
Obesity	13 (25.5%)	894 (24.8%)	.91
Unfit for open AAA or general anesthesia	12 (23.1%)	862 (23.9%)	.90

* Ankle-brachial index is missing in a considerable number of patients.

Results were reported as mean, range or standard deviation for continuous variables. Discrete variables were represented as proportions (%) of the study group. Preoperative patient characteristics, co-morbid factors, aneurysmal morphology at the time of the initial procedure, and details regarding the procedure and devices are correlated with the defined study groups by univariate analysis. Differences in findings between study groups were assessed by Chi-square tests for discrete variables and by Student's t and Mann–Whitney tests for continuous variables. A p-value $<.05$ was considered to represent a significant difference. Cumulative rates of freedom-from-aneurysm-related deaths, overall deaths, aneurysms rupture, conversion to open repair, endoleaks and increase of inflammatory reaction were assessed by life-table analysis. Significant differences between study groups were assessed by log-rank testing. Variables with clinical relevance were entered in a multivariate Cox-analysis to assess independent associations with late outcome. Postoperative change in aneurysm size in the IAAA group was compared to preoperative measurement by a paired T-test. All statistical analyses were performed with SAS Statistical Software (version 8.0, SAS Institute Inc., Cary, NC).

Results

The 3665 patients, 3461 male and 204 female, ranged in age from 43 to 95 years. Fifty-two patients (1.4%) had an IAAA, all diagnosed by CT, and 3613 (98.6%) a non-IAAA. The mean age in patients with IAAA was approximately 6 years less than in the other patients (Table 1). Other significant differences in patient characteristics included a higher incidence of smoking ($p=.0175$), and lower incidence of hypertension ($p=.0133$), better cardiac condition or less previous cardiac events ($p=.0272$) and less pulmonary disease ($p=.0032$) in the IAAA group. Regarding existing anatomy no differences were observed in angulation in the aneurysm neck ($p=.12$), the aneurysm itself ($p=.18$) or the iliac arteries ($p=.08$). The infrarenal neck was similar with regard to diameter ($p=.87$) and length ($p=.11$) in the two study groups. The aneurysm had comparable diameters ($p=.78$) and patency of iliac and hypogastric arteries.

Operating time was 140 min (45–345) in the group with IAAA compared to 133 min (25–660) in non-IAAA (ns). In the former group 47 had bifurcated endografts, one had a tube graft while four had aortouniiliac grafts. Extraanatomic bypasses were four times as frequent in the group with IAAA compared to non-IAAA (Table 2). Device or limb stenosis during the procedure occurred almost 18 times more frequently in the group with IAAA ($p=.0005$). Device migration as observed on the intraoperative angiogram did not occur in any of the patients with IAAA and in 40 of the patients with non-IAAA (1.1%). No differences were observed with regard to length of stay in hospital, prevalence of endoleak or the incidence of primary conversion to open surgery. Only blocking of one iliac artery was significantly different in the two study groups. Thirteen (25%) occurred in IAAA (nine intentional and four inadvertently) and 488 (13.5%) in non-IAAA, $p=.0100$.

Table 2. Procedural details and predischarge outcomes

	Inflammatory $N=52$	Non-inflammatory $N=3613$	p-value*
Failure to complete procedure	1 (1.9%)	58 (1.6%)	.69
Extra-anatomic bypass	2 (3.9%)	32 (0.9%)	.0086
Device related complications	6 (11.5%)	261 (7.2%)	.16
Device migration	0 (0.0%)	48 (1.3%)	–
Device/device limb stenosis	2 (3.9%)	9 (0.3%)	.0005
Hypogastric artery occlusion	13 (25.0%)	488 (13.5%)	.0100
Arterial complications	2 (3.9%)	128 (3.6%)	.72
Systemic complications	4 (7.7%)	438 (12.1%)	.63
Access site complications	2 (3.9%)	241 (6.7%)	.65
Type I endoleak	1 (1.9%)	158 (4.4%)	.50
Type II endoleak	6 (11.5%)	328 (9.1%)	.65
Type III endoleak	2 (3.9%)	88 (2.4%)	.62
Death $\leq$ 30 days	1 (1.9%)	81 (2.2%)	.66
Conversion $\leq$ 30 days	0 (0.0%)	42 (1.2%)	–
Rupture $\leq$ 30 days	0 (0.0%)	1 (0.03%)	–

* Adjusted for age, smoking, hypertension and cardiac and pulmonary risk status.

First-month outcome

The first-month mortality in the entire cohort was 2.2% (82 patients). There was no significant difference between the two study groups. There were no significant differences with regard to systemic complications (cardiac, cerebral, pulmonary, renal, hepatobiliary, bowel and sepsis) in the two study groups. Minor complications from the access sites and lower limb arteries were similar in the group with IAAA and non-IAAA (3.9 and 6.7%, respectively; ns). Arterial thrombosis occurred only in the group of patients with non-IAAA (0.8%). An increased periaortic inflammatory response was observed in 12% and a decreased periaortic inflammation in 17% of all patients with IAAA.

Late outcome

There were no differences in the incidence of type I, II and III endoleaks. The percentage of patients with aneurysmal growth was similar in the two groups. Device migration, kinking, stenosis or thrombosis was comparable in both groups. No differences were observed with regard to all-cause death, aneurysm-related death, rupture and conversion to open repair (Table 3). Of 47 patients with IAAA, diameter measurements were recorded during follow-up. A regression of the aneurysm was observed in 41 (87%, $p=.0001$) (Fig. 2). With regard to aneurysm shrinkage, no difference was observed between patients with and without IAAA.

Detailed information on 11 of the patients with IAAA

At presentation hydronephrosis was present in five patients (45%). Previous ureteric procedures had been performed in four patients (36%) (Table 4). Abdominal pain was present in 63% of the patients who had additional and detailed data provided by the questionnaire. Worsening of renal function in this subgroup was observed in the early postoperative period in 9% and in the late postoperative period in 27%. Postoperative ureteric stenting or ureterolysis was performed in two (18%) of these patients. No patients needed dialysis early or late postoperatively. Serum concentration of urea and creatinine decreased in these 11 patients, although not significantly. The ESR decreased during the early postoperative period. However, later it increased again to preoperative levels. The CRP levels decreased in the late postoperative phase compared to the preoperative phase. Aneurysm wall thickness decreased in the 11 patients with detailed information from 21 mm preoperatively to 17 mm early and 13 mm late postoperatively. Ureteric entrapment was observed in 45% of the patients preoperatively, decreasing to 27% after the procedure. In one patient the ureteric obstruction which was present preoperatively remained troublesome after operation with continued requirement for ureteric stenting.

Discussion

Taking into consideration that open surgery for IAAA is often challenging from a technical point of view with reported higher mortality and complication rate,[2,4,5] the present investigation indicates that with respect to exclusion of the aneurysm from the circulation, EVAR is a feasible method with promising early and midterm results. We have not observed a higher mortality or morbidity rate than for other aneurysms

Table 3. Late outcomes

Freedom of (4 years)	Inflammatory $N=52$ (%)	Non-inflammatory $N=3613$ (%)	p-value*
Type I endoleak	100.0	90.3	.97
Type II endoleak	77.8	83.8	.50
Type III endoleak	97.8	92.4	.86
Device migration	95.7	86.7	.59
Kinking	100.0	96.7	.99
Stenosis/thrombosis	97.9	94.5	.52
Aneurysm growth ≥ 8 mm	84.8	83.3	.22
Secondary endovascular intervention	83.4	88.8	.23
Death	92.8	81.6	.69
AAA-related death	98.1	96.2	.97
Conversion	95.7	94.3	.68
Rupture	100.0	98.6	.99

* Adjusted for age, smoking, hypertension and cardiac and pulmonary risk status.

in this study, which is in contrast with most reports on open surgery for IAAA. A mortality rate of 1.9% must be regarded as satisfactory, especially considering that 23.1% of the patients with IAA were unfit for open surgery. Systemic complications also were comparable.

While exclusion of the aneurysm seems to be obtained by EVAR in most cases, the effect on the fibrosis itself is less clear. Postoperatively both increased and decreased periaortic inflammation was observed on follow-up CT-scans although significant increase was only observed in six patients. The cause of this variable reaction regarding the fibrosis remains unknown. It is possible that the increased fibrosis in some cases could be related to the so-called 'post implantation reaction' occasionally seen in patients treated with EVAR. It would be of importance to follow these changes over the years, even if the renal function is not deteriorated. Following open operation, the fibrosis is decreasing in about 75% of the cases.[19,20] Although rare, increased fibrosis has also been reported following open surgery.[21,22] In patients with ureteral stenosis, regular CT-surveillance also seems indicated after open surgery.[22]

Even if the preoperative anatomy was similar in the two groups, there was an increased rate of graft limb stenosis in the IAAA group. As there were no significant anatomical differences between the two groups, the higher incidence of graft limb stenosis-occlusion may be related to the distal landing zone in the external iliac artery. This finding is associated with the more frequent overlapping of the hypogastric artery by the device limb in the IAAA group. One femorofemoral crossover was performed due to occlusion at the time of procedure. It is possible that the iliac arteries where encapsulated by fibrotic tissue and that the arterial wall as well as the aneurysm wall was stiffer than in patients with non-inflammatory aneurysms. Thus, modelling of the endoprosthesis with a balloon catheter after deployment could become more difficult. It is also possible that IAAA is a separate disease entity[23] with a higher incidence of autoimmune diseases[24] and a higher metabolic activity than non-inflammatory AAA.[25] However, although statistically significant, the total number of graft limb obstructions was small and further investigation of this particular phenomenon is necessary.

Hydronephrosis with or without ureteric procedures were frequently observed in the smaller subset with detailed information. It is likely that this

Table 4. Additional information on 11 patients with inflammatory aneurysms

	Preoperative	Early postoperative	Late postoperative
Clinical			
Worsening of renal function	5 (45%)	1 (9%)	3 (27%)
Ureter stent/urethrolysis	4 (36%)	1 (9%)	1 (9%)
Need for dialysis	–	0 (0%)	0 (0%)
Laboratory values	mean $\pm$ SD	mean $\pm$ SD	mean $\pm$ SD
Urea (mmol/l)	22.4 $\pm$ 21.7	10.0 $\pm$ 3.1	12.4 $\pm$ 6.2
Creatinine (µmol/l)	402 $\pm$ 537	118 $\pm$ 34	140 $\pm$ 56
ESR (mm/h)	50 $\pm$ 31	34 $\pm$ 35	45 $\pm$ 50
CRP (mg/l)	107 $\pm$ 81	66 $\pm$ 45	33 $\pm$ 45
CT findings			
Wall thickness (mm)	20.8 $\pm$ 15.9	17.4 $\pm$ 18.1	12.8 $\pm$ 18.4
Obvious decrease	–	6 (55%)	6 (55%)
Ureter entrapment	5 (45%)	4 (36%)	3 (27%)

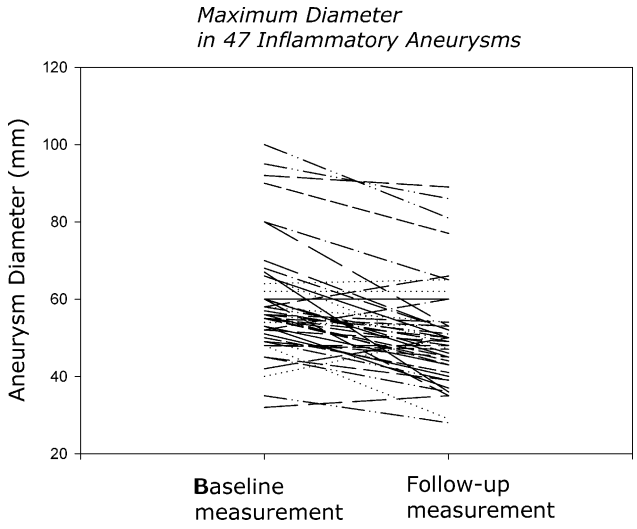

Fig. 2. Diameter changes in 47 patients with IAAA. The regression of aneurysm size in this group was significant ($p = .0001$).

was a selected group of patients with a high incidence of ureteric complications. Late postoperative worsening of renal function was present in 27%. These findings suggest that EVAR alone may not be the optimal treatment for all patients with IAAA. Possibly some patients with IAAA and ureteral stenosis might need post-EVAR ureterolysis, omental wrapping of the ureters or perhaps corticosteroid therapy, although this has not usually been considered necessary following open surgery. On the basis of the present analysis EVAR may especially be considered in patients with IAAA who have a high risk for open repair or in those who do not have ureteral stenosis. However, more studies are needed to determine whether EVAR is also the first-choice in the treatment of good-risk patients with IAAA.

The present study has several limitations including its retrospective nature and that it is based on questionnaires. It should be noted that the EUROSTAR database is not specifically designed for analysis of the typical pathology associated with IAAA. Nevertheless, we could identify a subgroup of patients with IAAA treated by EVAR, which is the largest series published in the literature so far. To assess some aspects of this typical condition in greater detail an additional questionnaire was mailed to the participants who had enrolled IAAA patients. This questionnaire resulted in more information in a proportion of our entire study group. The incidence of IAAA of 1.4% is lower than in most series treated with open surgery where an incidence of 5–10% has been reported.[5,6] Although unlikely, a lower incidence of IAAA in the present series could be explained by a smaller number of patients originally found suitable for EVAR according to the preoperative CT-scans or arteriograms, possibly due to the previously mentioned higher incidence of iliac aneurysms. It is also possible that more patients than we have detected in the registry so far, have an IAAA.

It should also be taken into consideration that about 23% of the patients were found unfit for open surgery and it is possible that some vascular surgeons did not find EVAR suitable treatment for patients with IAAA, especially in the early phase of our investigation. Thus, the group of patients unfit for open surgery perhaps consists of two subgroups; those considered unfit due to risk factors and those considered unfit because they had IAAA. This could explain why the incidence of patients unfit for open surgery were equal in the two groups, despite patients with IAAA being significantly younger. There are also differences in the classification of IAAA in patients treated by EVAR and those treated by open surgery. During open surgery the visual appearance of IAAA can be supplemented by a biopsy, whereas patients who are treated for EVAR must be classified according to the CT-scans only. Therefore, more inflammatory aneurysms may be identified during open surgery. Finding an IAAA by surprise is not rare. This may explain the relatively low incidence of IAAA in this study.

In conclusion, the results following EVAR of patients with IAAA and patients with non-IAAA were largely similar with regard to early and mid-term results. EVAR is a feasible method to exclude IAAA from the circulation. Perianeurysmal fibrosis did not regress in a proportion of patients, however, clinical outcome was favourable. The effect upon the fibrotic changes needs to be studied more thoroughly especially in patients with ureteric complications to define the exact role of EVAR in patients with inflammatory aortic aneurysms.

Acknowledgements

The EUROSTAR Collaborative centers are:
Belgium: Aalst, ASZ; Aalst, Onze Lieve Vrouwe Hospital; Antwerpen, AZ Middelheim; Antwerpen, St Augustinus Hospital; Assebroek, AZ St Lucas/St Jozef; Bonheiden, Imelda Hospital; Brussels, University Hospital St Luc; Genk, St Jan Hospital; Gent, Volkskliniek; Gent, AZ St Lucas; Gilly, St Jospeh Hospital; Kortrijk, AZ Groenige; Leuven, University Hospital; Liège, University Hospital; Lommel, Maria Hospital; Mechelen, OLV Hospital; Mouscron, CHM CNDT; St Truiden, St Trudo Hospital; Vilvoorde, St Josef Hospital.
Denmark: Copenhagen, Rigshospitalet; Odense, University Hospital.

France: Draguignan, Hospital Notre Dame; Paris, Hospital Henri Mondor.
Germany: Dusseldorf, Augusta Hospital; Frankfurt, Städtischen Kliniken; Frankfurt, Sankt Katharinen; Hamburg, Altona General Hospital; Kempten, Klinikum Kempten; Koblenz, Bundeswehrzentral; Marburg, Philipps-University; Oldenburg, Pius Hospital.
Israel: Tel Aviv, Sheba Medical Centre.
Italy: Perugia, Policlinico Monteluce; Roma, Ospedale S Giovanni.
Luxembourg: Luxembourg, Centre Hospitalier.
Monaco: Monaco, Centre Cardio-Thoracique.
The Netherlands: Amsterdam, Academic Medical Centre; Amsterdam, VU Hospital; Amsterdam, OLV Gasthuis; Apeldoorn, Gelre Hospital; Arnhem, Rijnstate; Breda, Amphia Hospital; Delft, Reinier de Graaf Group; Dordrecht, Albert Schweitzer Hospital; Eindhoven, Catharina Hospital; Enschede, Medisch Spectrum Twente; Geldrop, St Anna Hospital; Groningen; Academic Hospital; Groningen, Martini Hospital; Maastricht, Academic Hospital; Nieuwegein, St Antonius Hospital; Nijmegen, CWZ Hospital; Nijmegen, Academic Hospital St Radboud; Rotterdam, St Clara Hospital; Rotterdam, Dijkzicht Hospital; Rotterdam, Franciscus Gasthuis; The Hague, Medical Centre Haaglanden Westeinde; The Hague, Leijenburg Hospital; Tilburg, Tweesteden Hospital; Utrecht, University Medical Centre; Veldhoven, St Josef Hospital; Zwolle, Isala Clinics Sophia.
Norway: Oslo, Aker University Hospital; Oslo, Ulleval Hospital; Trondheim, University Hospital.
Poland: Lublin, L'Academie de medicine; Warsaw, Medical University.
Spain: Barcelona, University Hospital; Barcelona, Ciutat Sanitaria I Universitaria de Bellvitge; Barcelona, Hospital Santa Creu I S Pau; Donostia San Sebastian, Hospital de Gipuzkoa; La Coruña, Hospital Juan Canalejo; Lugo, Hospital Xeral Lugo; Madrid, University Hospital de la Princesa; Madrid, Hospital Ramon y Cajal; Madrid, Fundacion Jimenez Diaz; Madrid, University Hospital of Getafe; Pamplona, University Hospital of Navarra; Valladolid, Hospital Clinico Valladolid.
Sweden: Lund, University Hospital; Örebro, Medical Centre; Stockholm, Karolinska Hospital.
Switzerland: Bern, Clinic for Cardiovascular Surgery; Zürich, Gefasszentrum.
United Kingdom: Bournemouth, Royal Hospital; Bristol, Royal Infirmary; Chester, Countess of Chester Hospital; Glasgow, Gartnavel Hospital; Liverpool, Royal University Hospital; Newcastle-Upon-Tyne, Freeman Hospital.

References

1 Walker DI, Bloor K, Williams G, Gillie I. Inflammatory aneurysms of the abdominal aorta. *Br J Surg* 1972;**59**:609–614.
2 Crawford JL, Stowe CL, Safi HJ, Hallman CH, Crawford ES. Inflammatory aneurysms of the aorta. *J Vasc Surg* 1985;**2**:113–124.
3 Rasmussen TE, Hallett Jr JW. New insights into inflammatory abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 1997; **14**:329–332.
4 Lacquet JP, Lacroix H, Nevelsteen A, Suy R. Inflammatory abdominal aortic aneurysms: a retrospective study of 110 cases. *Acta Chir Belg* 1997;**97**:286–292.
5 Pennell RC, Hollier LH, Lie JT, Bernatz PE, Joyce JW, Pairolero PC *et al*. Inflammatory abdominal aortic aneurysms: a thirty-year review. *J Vasc Surg* 1985;**2**:859–869.
6 Lindblad B, Almgren B, Bergqvist D, Eriksson I, Forsberg O, Glimåker H *et al*. Abdominal aortic aneurysm with perianeurysmal fibrosis: experience from 11 Swedish vascular centers. *J Vasc Surg* 1991;**13**:231–239.
7 Boyle JR, Thompson MM, Nasim A, Sayers RD, Holmes M, Bell PRF. Endovascular repair of an inflammatory aortic aneurysm. *Eur J Vasc Endovasc Surg* 1997;**13**:328–329.
8 Chuter T, Ivancev K, Malina M, Lindblad B, Brunkwall J, Risberg B. Inflammatory aneurysm treated by means of transfemoral endovascular graft insertion. *J Vasc Interv Radiol* 1997;**8**:39–41.
9 Deleersnijder R, Daenens K, Fourneau I, Maleux G, Nevelsteen A. Endovascular repair of inflammatory abdominal aortic aneurysms with special reference to concomitant ureteric obstruction. *Eur J Vasc Endovasc Surg* 2002;**24**:146–149.
10 Hinchliffe RJ, Macierewicz JA, Hopkinson BR. Endovascular repair of inflammatory abdominal aortic aneurysms. *J Endovasc Ther* 2002;**9**:277–281.
11 Vallabhaneni SR, Mc Williams RG, Anbarasu A, Rowlands PC, Brennan JA, Gould DA *et al*. Perianeurysmal fibrosis: a relative contra-indication to endovascular repair. *Eur J Vasc Endovasc Surg* 2001;**22**:535–541.
12 Barrett JA, Wells IP, Roobottom CA, Ashley A. Progression of peri-aortic fibrosis despite endovascular repair of an inflammatory aneurysm. *Eur J Vasc Endovasc Surg* 2001;**21**:567–568.
13 Harris PL, Buth J, Mialhe C, Myhre HO, Norgren L. The need for clinical trials of endovascular abdominal aortic aneurysm stent-graft repair: the EUROSTAR project. EUROpean collaborators on stent-graft techniques for abdominal aortic aneurysm repair. *J Endovasc Surg* 1997;**4**:72–77.
14 Buth J. Endovascular repair of abdominal aortic aneurysms. Results from the EUROSTAR registry. EUROpean collaborators on stent-grafts techniques for abdominal aortic aneurysm repair. *Semin Interv Cardiol* 2000;**5**:29–33.
15 Buth J, Laheij RJ. Early complications and endoleaks after endovascular abdominal aortic aneurysm repair: report of a multicenter study. *J Vasc Surg* 2000;**31**:134–146.
16 Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM *et al*. Reporting standards for endovascular aortic aneurysm repair. *J Vasc Surg* 2002;**35**:1048–1060.
17 Arko FR, Anthony Lee W, Hill BB, Olcott C, Dalman RL, Harris EJ *et al*. Aneurysm-related death: primary endpoint analysis for comparison of open and endovascular repair. *J Vasc Surg* 2002;**36**:297–304.
18 White GH, Yu W, May J, Chaufour X, Stephen MS. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis and management. *J Endovasc Surg* 1997;**4**:152–168.
19 Bitsch M, Nørgaard HH, Røder O, Schroeder TV, Lorentzen JE. Inflammatory aortic aneurysms: regression of fibrosis after aneurysm surgery. *Eur J Vasc Endovasc Surg* 1997; **13**:371–374.
20 Stella A, Gargiulo M, Faggioli GL, Bertoni F, Cappello I, Brusori S *et al*. Postoperative course of inflammatory abdominal aortic aneurysms. *Ann Vasc Surg* 1993;**7**:229–238.
21 Stotter AT, Grigg MJ, Mansfield AO. The response of perianeurysmal fibrosis—the 'inflammatory' aneurysm—to surgery and steroid therapy. *Eur J Vasc Surg* 1990;**4**:201–205.
22 von Fritschen U, Malzfeld E, Clasen A, Kortmann H. Inflammatory abdominal aortic aneurysm: a postoperative course of retroperitoneal fibrosis. *J Vasc Surg* 1999;**30**:1090–1098.
23 Rasmussen TE, Hallett JW, Mathieu Metzger RL, Richardson DM, Harmsen WS, Goronzy JJ *et al*. Genetic risk factors in inflammatory abdominal aortic aneurysms:

plymorphic residue 70 in the HLA-DR B1 gene as a key genetic element. *J Vasc Surg* 1997;**25**:356–364.
24 Haug E, Skomsvoll JF, Jacobsen G, Halvorsen T, Sæther OD, Myhre HO. Inflammatory aortic aneurysm is associated with increased incidence of autoimmune disease. *J Vasc Surg* 2003; **38**:492–497.
25 Tennant WG, Baird RN, Horrocks M. Metabolic Activity in inflammatory and non-inflammatory aneurysms of the abdominal aorta. *Eur J Vasc Surg* 1992;**6**:199–203.

Accepted 4 January 2005
Available online 2 February 2005